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TI Store depletion triggers the calcium release-activated calcium current (ICRAC) in macrovascular endothelial cells: a comparison with Jurkat and embryonic kidney cell lines.

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AB In endothelial cells, different types of Ca^{2+} conductances have been described, but none of them has been clearly identified as ICRAC, the

Ca^{2+}

release-activated Ca^{2+} current originally described in mast and lymphoma cells. Here we show that in bovine pulmonary artery endothelial cells (CPAE) depletion of intracellular Ca^{2+} stores by inositol 1,4,5-trisphosphate (InsP_3), Ca^{2+} ionophores and Ca^{2+} pump inhibitors activates a Ca^{2+} -selective conductance in the presence of the Ca^{2+} chelator 1,2-bis(2-aminophenoxy)ethane- $\text{N},\text{N},\text{N}',\text{N}'$ -tetraacetic acid (BAPTA). The current shows inward rectification, a highly positive reversal potential and is blocked by micromolar concentrations of La^{3+} . The conditions used in studies of endothelial cells were also employed in those of HEK-293, an **embryonic** kidney cell line commonly used to express putative store-operated channels, and Jurkat cells, the **reference cell** model. Similar to CPAE, HEK cells also have an ICRAC-like current. At 0 mV

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L1	728 S REFERENCE (W) CELL?
L2	11 S L1 (P) EMBRYO?
L3	0 S L2 (P) (ADVANTAG? OR USEFUL? OR PREFERR?)
L4	5 DUPLICATE REMOVE L2 (6 DUPLICATES REMOVED)